An Acute Linear Pruritic Eruption Following Allergic Contact Dermatitis

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Case Report

A 39-year-old man presented to the emergency department with an abscess in the left axilla that was treated with incision and drainage and a 10-day course of trimethoprim/sulfamethoxazole. Two days later, he reported worsening erythema and tenderness at the abscess site. While changing the wound dressing, he noticed that the skin under the adhesive tape was red and irritated. Concerned about an allergic reaction to the adhesive tape, he avoided further use. The following day, he developed an acute, symmetric, pruritic eruption in previously unaffected skin on the arms and hands. He was otherwise well without systemic complaints. He had previously tolerated trimethoprim/sulfamethoxazole without any adverse effects and had no prior history of eczema or other dermatitis.

Physical examination revealed a tender, erythematous, fluctuant cyst in the mid left axilla. Geometric erythema and a superficial erosion were most prominent medial to the cyst at the site of the applied adhesive tape (Figure 1). The forearms and dorsal hands demonstrated numerous 0.1 to 0.2cm nonfollicular, clear-to-white papules and papulovesicles (Figure 2), many of which were arranged in a linear distribution (Figure 3). The trunk, legs, and feet were uninvolved. Results of a complete blood cell count and basic metabolic panel were unremarkable. Bacterial culture of the left axillary abscess demonstrated few methicillin-resistant *Staphylococcus aureus*. A biopsy of the linear lesions on the right forearm was performed for histopathologic examination (Figure 4).

Diagnosis

Koebnerizing id reaction

Microscopic Findings and Clinical Course

Histopathologic examination of the skin biopsy demonstrated a spongiotic dermatitis with a perivascular lymphohistiocytic infiltrate. A focal intraepidermal vesicle containing neutrophils and lymphocytes was present (Figure 4). The patient was treated symptomatically with 25mg hydroxyzine orally every six hours as needed and 0.1% triamcinolone cream twice a day. On follow-up telephone conversation at five weeks, all lesions had resolved.

Discussion

Originally described in 1876 by Henrich Koebner to explain the appearance of psoriatic lesions appearing in areas of uninvolved skin after trauma, the isomorphic, or Koebner response, has now been identified in a variety of dermatological conditions.[1] Further classification of Koebnerization by Boyd and Neldner[2] separates the reactions by criteria such as pathogenesis, frequency, and reproducibility into the following four classes: 1) true isomorphic response including lichen planus, psoriasis, and vitiligo; 2) pseudo-isomorphic response including Behçet's disease, molluscum contagiosum, pyoderma gangrenosum, and warts; 3) occasional traumatic localization of lesions including Darier disease, erythema multiforme, Hailey-Hailey disease, Kaposi sarcoma, and lichen sclerosus; and 4) poor or questionable trauma-induced processes including bullous pemphigoid, discoid lupus erythematosus, eczema, lichen nitidus, and nevocytic nevi. The timing of onset between injury to uninvolved skin and the appearance of disease is usually 10 to 14 days, but may be as short as three days or as long as several years.[2]

The id reaction, also known as autoeczematization or autosensitization dermatitis, refers to the sudden development of dermatitis at a distant site from a local inflammatory reaction. A variety of stimuli have been reported to cause id reactions including fungal (e.g., dermatophytosis, candidiasis, and coccidiomycosis), bacterial (e.g., tuberculosis and leprosy), viral (e.g., herpes simplex virus and poxvirus),

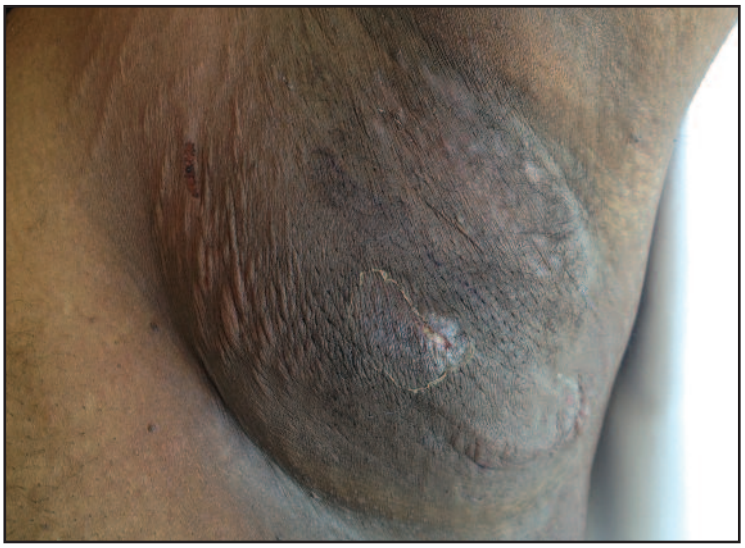

Figure 1. A tender, erythematous, fluctuant cyst is present in the mid left axilla. Geometric erythema and a superficial erosion is noted most prominently medial to the cyst at the site of the applied adhesive tape.

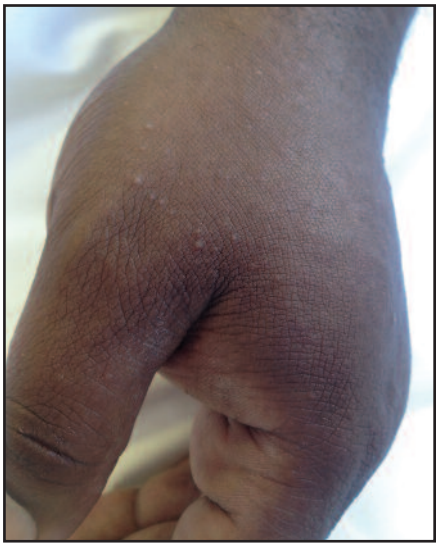

Figure 2. Numerous pruritic, non-follicular, clear-to-white papulovesicles on the dorsal hand

and parasitic (e.g., leishmaniasis, lice, and scabies) skin infections as well as allergic contact dermatitis (e.g., nickel). Most commonly, id reactions are attributed to superficial dermatophytoses, especially tinea pedis. Classically, an acute, intensely pruritic, symmetric, maculopapular or papulovesicular eruption occurs, usually involving the extremities. While some authorities have broadened the definition of id reactions to include hypersensitivity reactions, such as erythema nodosum, erythema multiforme, Sweet's syndrome, migratory thrombophlebitis, erysipelas-like dermatitis, and urticaria,[3] the authors prefer to limit the definition to eczematous processes (autoeczematization) such as that observed in this case. Lesions commonly appear within days to weeks of the primary inflammation. Histology is nonspecific, often demonstrating a vesicular spongiotic dermatitis in which eosinophils may be present in the infiltrate. Systemic symptoms including fever, anorexia, lymphadenopathy, splenomegaly, arthralgias, and hematologic abnormalities have been reported in association with id reactions.[3] Treatment of id reactions is aimed at treating the inciting infection or dermatitis, after which the eruption resolves. Symptomatic treatment of pruritic eruptions can be achieved with systemic or topical corticosteroids and antihistamines.

The pathogenesis of these entities remains elusive, yet both are seemingly related to a decrease in the skin's threshold for irritation due to immune responses.[4–6] A proliferation of activated T cells is presumed to be pathogenic in autoeczematization reactions.[5] A two-step model has been proposed for the pathogenesis of the Koebner phenomenon—a first, nonspecific inflammatory step contributes to the production of cytokines, stress proteins, adhesion molecules, or autoantigens translocated from intracellular areas. The second step may involve disease-specific reactions, induced by T cells, B cells, autoantibodies, and immune deposits in genetically predisposed patients.[6]

The authors' case is a unique presentation of an id reaction in that Koebnerization is a prominent feature. It is plausible that the etiology of their patient's id reaction could be attributed in whole or in part to the allergic contact dermatitis and infectious abscess as both have been associated with id reactions.[6,7] The differential diagnosis included lichen niditus and eczema, each of which may Koebnerize; however, the close proximity to the time of onset of the eruption with the allergic contact dermatitis and the histopathology are consistent with an id reaction. Additionally, the absence of a prior history of lichen nitidus and the rapid resolution of the papules with triamcinolone cream further support the diagnosis of an id reaction over lichen nitidus.

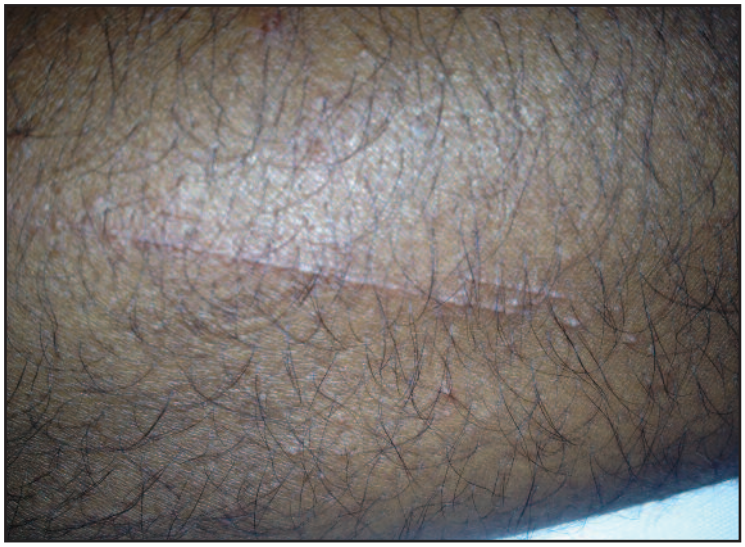

Figure 3. On the right forearm, the papulovesicles were arranged in a linear distribution.

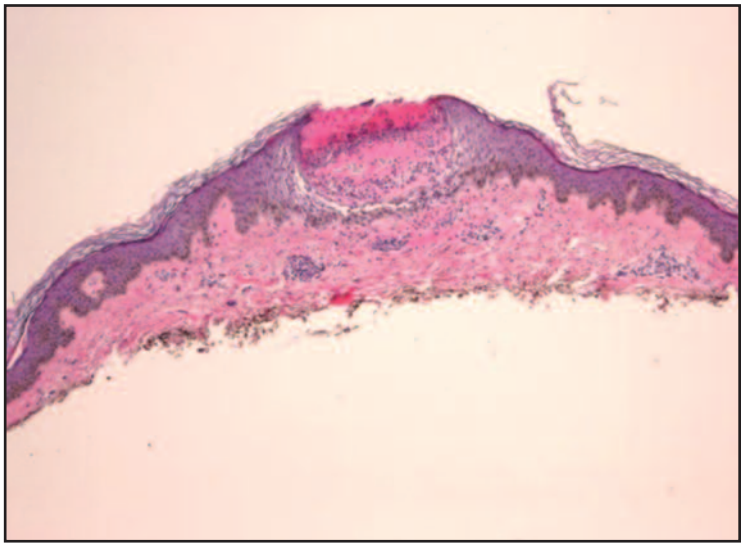

Figure 4. Histopathologic examination of the right forearm demonstrated a spongiotic dermatitis with a perivascular lymphohistiocytic infiltrate. A focal intraepidermal vesicle containing neutrophils and lymphocytes was present (H&E 40x).

To the authors' knowledge, this is the first case reported to include both the id and Koebner phenomenon. According to the Boyd-Neldner classification of the Koebner phenomenon, the id reaction would likely be classified as poor or questionable trauma-induced process as there have been no known previous occurrences, despite clear Koebnerization in this case.[2] When a patient presents with an acute, widespread, and symmetric eruption, it behooves the clinician to consider an id reaction in the differential and carefully examine the patient for a distant inflammatory process or skin infection. Treatment of the underlying condition is essential for resolution of the id reaction.

References

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